The First Open Heart Corrections of Tetralogy of Fallot

A 26–31 Year Follow-up of 106 Patients

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Tetralogy of Fallot became a correctable malformation on August 31, 1954, and from that date through 1960, 106 patients (ages 4 months–45 years) who underwent open repairs at the University of Minnesota and were discharged, have been followed (99% complete) until death or for 26–31 years (mean: 23.7 years, 2424 patient years). The purposes of this study were to determine survival, morbidity, hemodynamics, educational/employment attainments, and relation of these to surgical technics. Operations were done by cross circulation (6 patients) and bubble oxygenator (100 patients). This group had the first uses of patch ventricular septal defect closure, outflow root, infundibuloplasty, atresia correction, ischemic arrests, and pacemakers among other innovations. Twenty-one (of 105 patients) have died during the followup: eight deaths in the first 10 years, 12 between 10 and 20 years, and 1 >20 years. The causes of death were sudden (5), accidental (4), congestive failure (2), reoperation (2), suicide (2), and other (2). Actuarial survival at 30 years was 77%. Late complications were ten reoperations, five arrhythmias, and one endocarditis. Actuarial freedom from reoperations at 30 years was 91%. Cardiac recatheterizations in 62 patients disclosed only 10 with residual shunts. Peak right ventricular systolic pressures were <40 mmHg (34 patients), 41–60 mm (2 patients), 61–70 mm (4 patients), >71 mm (4 patients). Thirty-four patients (32%) completed college, ten of these completed graduate school (5 masters degrees, 2 M.D.'s, 2 Ph.D.'s, 1 lawyer). Fifteen others attended college, and nine received technical school diplomas. Forty patients (18 men, 22 women) had progeny, with 82 (93%) live births and six major cardiac defects (7.3%). In summary, complete repair gave excellent late results in this group cared for very early in the open heart era. Survivors led productive lives without restrictions in education and employment. Many of the deaths/complications that occurred are now easily preventable, which augurs extremely well for this generation.

Second thoughts are ever wiser

from Euripides, 4th Century, B.C.

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TETRALOGY OF FALLOT (TOF), a common form of cyanotic heart disease in infants and children, became a correctable malformation on August 31, 1954, when open cardiotomy with extracorporeal circulation by controlled cross-circulation was utilized. The first operation by these methods was successful, and the patient is living an active, normal life as a professional musician 31.5 years after operation.[1]

The corrective procedures consisted of closing the ventricular septal defect and relieving the pulmonary stenosis. The intracardiac operation was, at that time, a significant departure from the palliation afforded by systemic–pulmonary artery shunts and thus provoked considerable discussion at the time of our first report of a series of ten patients in April 1955.[2] As noted in that presentation, we were sufficiently impressed by the well being of those early patients to make the intracardiac corrective operation our method of choice, with very few exceptions, for all TOF patients referred for surgical treatment from that time on.

The purposes of this study were to determine survival, morbidity, hemodynamics, child bearing, educational and employment attainments, and the relation of these to surgical techniques. Operations were done by controlled cross circulation[3–5] (6 patients) and DeWall–Lillehei bubble oxygenator[6–8] (100 patients). This patient group had the first uses of patch ventricular septal defect (VSD) closure, outflow roof, infundibuloplasty, atresia correction, ischemic arrest, and pacemakers among other innovations.

Patients and Methods

The patients reported herein were all of those who had intracardiac repair of their TOF defects from August 31,

1954, through December 31, 1960, at the University of Minnesota and Variety Club Hospital, and who were discharged from the hospital after their operation.

The patients' ages at the time of operation ranged from 4 months to 45 years (Table 1). Both the youngest operated on as an emergency (now 31½ years old and a general surgeon) and the oldest (a grandmother now 75 years of age) are long-time survivors currently living active normal lives.

All patients have been followed either until death or for a minimum of 26 years (to December 31, 1985) and a maximum of 31½ years.

A total of 2424 patient years were observed after operation, and the mean period of follow-up was 23.7 years.

The hospital mortality, while not a part of this study, ranged from four patients (40%) for the first ten patients operated on in 1954 and early 1955[2] to 14.9% for those operated on in 1960.

Definition of Tetralogy of Fallot

All patients reported in this study had a cardiac malformation in which there was a ventricular septal defect, as defined below, and a pulmonary stenosis with specific characteristics.

The septal defect was large, that is, of a size approximating that of the aortic orifice. Its location was nearly always high, *i.e.*, close beneath the aortic valve.

All patients had an infundibular pulmonary stenosis. More than one half had an additional valvar stenosis. A much smaller number had complete atresia of the right ventricular outflow tract. The pulmonary stenosis was sufficiently severe that the peak pressures in the two ventricules were similar at rest or with exercise.

Patients with this general malformation who had mild pulmonary stenosis were not included in our series as TOF. Also not included were several patients with severe infundibular pulmonic stenoses associated with very small ventricular defects (<5 mm). Both of these malformations differ from TOF in their clinical manifestations and also present simpler lesions for surgical treatment.

Patient Follow-up

As noted, 106 patients comprised the entire group, and direct contact has been established with 105 (99%) by one or more of the following methods: personal interview and examination, a detailed questionnaire filled out by the patient, telephone interview with patient supplemented by questionnaires, reports from the patients' personal physicians. Death information was obtained from the family and was supplemented in 100% of the patients by reports from the physicians in attendance. Autopsy information was available for 12 of the 21 late deaths (57%).

TABLE 1. *Tetralogy of Fallot—26–31 Year Follow-up*

Age at Operation	Patients (N)
Under 2 yr (youngest 4 mo)	11 (10.4%)
2–5 yrs.	18
5–15 yr	63
Over 15 yr (oldest 45 yr)	14 (13.2%)
Total	106

In approximately three fourths of these patients, contact had been maintained over the years by annual reports.

Operative Procedures and Technics

In the beginning, corrective surgical treatment for TOF patients faced two formidable problems.

First was the need to develop methods that would permit intracardiac exposure of the cardiac interior in a relatively bloodless field for a sufficient length of time to repair technically complex malformations. The first six patients in this study, operated on between August 31, 1954, and April 12, 1955, had extracorporeal circulation by controlled cross circulation.[3–5] A 30-year follow-up report on all these patients has been made recently.[1] The subsequent 100 patients all had extracorporeal circulation utilizing the DeWall–Lillehei bubble oxygenator.[6–8]

Second was the need to learn to recognize the extremely wide spectrum of pathologic anatomy comprising this diagnostic category and to devise methods for satisfactory repair.

Some of the major technical milestones that proved so valuable in achieving effective results are worthy of brief mention here as they illustrate very well the evolution of the technical innovations that have had a profound influence on operative and long-term survival.

Patch Closure of Ventricular Septal Defects

By April 1955, we had realized that the ventricular defects of tetralogy were typically larger and more muscular than other ventricular defects, so that stitches with a patch of synthetic material began to be used.[2] This technique reduced the risks of incomplete closure and injuries to the conduction system.

Ischemic Cardiac Arrest

Also, in the earliest tetralogies operated on in 1954–1955, selective aortic occlusion was introduced and found valuable for improving visualization without harm to the myocardium.[2,4]

Pulmonary Atresia Correction

One of the patients operated on in the first series of ten patients was an infant who unexpectedly had a complete

atresia of the origin of the pulmonary artery. We were able successfully to anastomose the main pulmonary artery directly to the newly constructed outflow trail of the right ventricle.

The ability to deal with this more complicated variety of tetralogy was very reassuring at the time. The technique that was used was successful in several other patients in 1955, and its applicability was further broadened by the introduction of the concept of the outflow roof in 1956.

Right Ventricular Outflow Roof: Subanular or Transanular

The introduction of this concept immediately improved significantly the surgeon's ability to deal effectively with the severe degrees of hypoplasia that are often a part of the tetralogy malformation. It has proved enormously effective in dealing immediately with the very severely stenotic outflow tracts associated with hypoplasia, but also the long range results have been good because of the growth that can occur under the roof. When it has been properly used, there have been very few deleterious effects on the myocardium over the many years these patients have now been observed.

The lack of serious long-term effects on the myocardium from valvular insufficiency is quite understandable in the light of the physiological studies on myocardial work done by Sarnoff and coworkers.[9] The latter showed that approximately 80–85% of ventricular work is pressure work (static energy), necessary to open the outlet valve, and only about 15–20% is volume work (kinetic energy). Sarnoff's group[9] and Braunwald[26] have showed that ventricular stroke volumes (kinetic energy) can increase significantly, even double, with only barely measurable increases in overall ventricular work (oxygen consumption).

Since regurgitation increases stroke volume, which adds to the kinetic energy fraction, it is clear why even quite sizeable amounts of regurgitation have only a small overall effect on ventricular work.

This then is the physiologic basis for what clinicians have noted over many years in these patients, namely, that the regurgitation that may be induced by an outflow roof, even if transanular, is very well tolerated. The counterpart is that relief of pressure work (obstruction) is very important to the myocardial integrity.

The initial use of an outflow roof on April 27, 1956, was transanular and was a matter of expediency.

The patient had had a closed Brock-type procedure as an emergency at the age of 3 years in 1952. Some temporary palliation was achieved, but by 6 years of age he needed help again. After he was put on cardiopulmonary bypass, his ventricular defect was closed, and we were faced with a small main pulmonary artery with a very firm fibrous ring at its origin. There was no pulmonary valve visible. We were unable to dilate this ring effectively, and there was no tissue to excise. Hence, the artery and ring were laid open, and a transanular patch of compressed polyvinyl sponge was sewn into the outflow tract. We were impressed at the time that his recovery was so uneventful. (This first patient is a 30-year survivor with no exercise limitations whatsoever.)

Infundibuloplasty

This valuable technique for widening the outflow track evolved gradually over several years and became the mainstay of the methods at the surgeon's disposal for relief of pulmonic stenosis.

Infundibuloplasty creates a widening of the right ventricular outflow track by mobilizing the crista supraventricularis (CS) away from the anterior ventricular wall. This allows the CS to drop down from the anterior ventricular wall, thereby enlarging the ventricular outflow passage. At the same time, this uncoupling of the CS displaces it downward as a flap toward the ventricular septal defect, aiding both in the exposure and closure of the latter. The technical details for infundibuloplasty have been described previously.[10,11]

Infundibuloplasty is applicable in most patients with TOF and is done first. Then excision of obstructing muscle tissue, if feasible, is performed, and finally an outflow roof is utilized as deemed necessary.

Complete Heart Block and the Development of Pacemaker Treatment

An unexpected bonus from the development of open heart surgery was the discovery of a revolutionary new concept for the treatment of complete heart block by use of myocardial electrodes and a pulse generator (pacemaker). This treatment dramatically reduced the high hospital mortality from complete heart block that occurred prior to the first clinical use of this new method on our service on January 30, 1957. Obviously, this method has had enormous value extending far beyond open heart surgery patients.

In the early intracardiac procedures for ventricular septal defects, tetralogy, or atrioventricular canal defects performed during 1954–1955, permanent complete heart block occurred in about 10% of the operations (7 patients out of the first 70 operations). With the then existing treatments for complete block by positive chronotropic drugs (epinephrine, ephedrine), atropine, or electrodes applied to the surface of the chest, there were no postoperative survivors, all died in the hospital before discharge.[1,12]

After we started use of the new drug isoproterenol (Isuprel®)* in August 1955, there were seven out of 13 pa-

* Developed for the treatment of bronchospasm, it was found to have potent positive ionotropic and chronotropic effects on the heart without appreciable effects on the blood pressure. These actions made it the most effective drug then available for the treatment of complete heart block.

tients with complete block (54%) who were postoperative survivors and were discharged from the hospital.[12,13]

Spurred by these adversities and the still high mortality, even with Isuprel, the problem was taken to the experimental laboratory where Lillehei and Weirich[14,15] found in 1956 that a myocardial electrode connected to a pulse generator producing repetitive electrical stimuli of small magnitude (5–10 milliamps) gave very effective control of the heart rate and was imperceptible to the animals and patients.

After clinical treatment of complete heart block with myocardial electrodes and external pacemaker was instituted in January 1957, 17 of the next 19 patients (89%) were hospital survivors.[12] It was found that when patients could be kept alive with a pacemaker, about 50% or more reverted to sinus rhythm, but this reversion rarely occurred after 30 days.

The next very important lesson took several more years to learn, namely, that the late mortality rate in postsurgical chronic complete heart block patients was extremely high even though most patients clinically appeared to be doing very well until their sudden fatal Adams–Stokes attack.[16]

Several of the late deaths in this series were part of that learning experience. The evidence became conclusive that all patients in complete heart block must have permanent pacing. This latter became eminently more practical with the development of an effective implantable unit by Chardack and Greatbach.[17]

Results

An overview of this patient group is provided in Table 2. Of particular interest in this follow-up study are the causes of late death and the quality of life of the long-time survivors.

Survival and Late Mortality

In the 26–31 year period since these 105 patients were discharged from the hospital, there have been 21 deaths (20%). Their causes and times after operation have been detailed in Table 3.

Their actuarial survivals were 92.5% at 10 years, 80% at 20 years, and 77% at 30 years. (Fig. 1). Of the 21 deaths, 13 (62%) were clearly cardiac in origin and, interestingly, eight (38%) were from causes (trauma, suicide, or cancer) that appeared unrelated to either their birth malformation or their subsequent treatment.

The commonest cause of late death (5 patients, Table 3) was a sudden and unexpected event in patients who appeared to be doing well. These all occurred beyond the first decade after operation (Table 3), adding further circumstantial evidence for arrhythmias as a primary cause. However, a very careful analysis of all of our operative and postoperative data on these particular patients has failed even in retrospect to uncover any common etiologic factors. The possible sudden onset of complete heart block remains a theoretical possibility, but there has been little supporting evidence for this mechanism.

TABLE 2. *Corrective Operations for Tetralogy of Fallot—26–31 Year Follow-up*

Total no. discharged after operation (1954 through 1960)	106 patients
Follow-up complete	105 (99%)
Alive (1986)	84 (80%)
Late deaths	21 (20%)

Total follow-Up: 2424 patient years (mean: 23.7 yr)

Of the five patients in the group, two had had a temporary period of complete heart block at the time of operation:

> One, a 22-month-old infant operated on in 1955 before the development of the pacemaker, had this arrhythmia noted for 10 minutes immediately postpump. Then sinus rhythm resumed. His sudden death occurred 14 years later.
>
> The other patient, 10 years old at open operation in 1959 (a closed Brock operation had been done in 1952), had pacemaker treatment for 1 week before sinus rhythm resumed. Sudden death was 19 years later.

However, many patients who are doing well today had similar histories.

The difficulty of proving arrhythmias as the cause of these deaths, by conventional data, is well exemplified by two other sudden-death patients who had very complete follow-up information, including normal postoperative heart catheterization and complete autopsies.

> One was a man 18 years of age in 1958 at the time of correction by patch closure of his ventricular defect and infundibulectomy. One year after operation he had had a near normal right heart catheterization with no shunts and an outflow gradient of 9 mmHg. In the years following surgery he had obtained a Ph.D. degree (Divinity). He was found dead in his office 27 years after operation at age 45 years. At autopsy a definite cause of death was not identified. A moderately severe degree of coronary artery disease was found,

TABLE 3. *Late Deaths*

Causes	Years Postoperative 10	10–20	20	Totals
Sudden/unexpected (arrthymias?)	—	4	1	5
Complete heart blocks	4	—	—	4
Accidents	1	3	—	4
Suicide	—	2	—	2
Reoperations	1	1	—	2
Heart failure	2	—	—	2
Brain tumor	—	1	—	1
Murdered	—	1	—	1
Totals	8	12	1	21

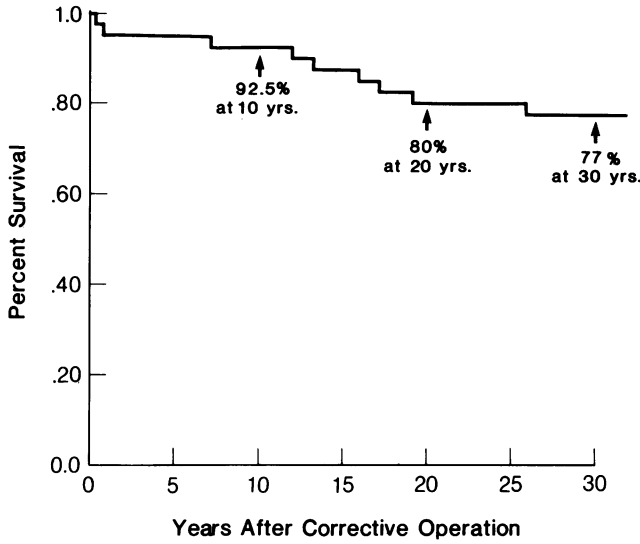

FIG. 1. Actuarial survival of 105 patients discharged from the hospital after repair of tetralogy of Fallot defects.

but no acute changes were seen. Review of his operative notes indicated no episodes of even transitory heart block. During his immediate hospital convalescence, Pronestyl® was administered for tachycardia (of unspecified nature). This was discontinued before discharge.

The other patient was a boy 9 years of age when his ventricular septal defect was closed by primary suture and an infundibulectomy was performed. There were no abnormal arrhythmias, even transitory in the operating room or after operation. One year after operation he had a completely normal right heart catheterization without shunt or pulmonary gradient. His right ventricular pressure was 28/0 mmHg. In July 1970, at age 21 years, he was brought to the emergency room of the local hospital. His heart was in ventricular fibrillation, and there was no satisfactory response to resuscitation. He had been working as an automobile mechanic and had no restrictions of any kind on his physical activities. For several weeks prior to death, he had complained of dizzy spells but did not seek medical attention. At autopsy, including gross and microscopic examination of the brain, a cause of death was not found. There was no residual pulmonary stenosis nor septal defect. The area of the VSD closure was calcified. The heart specimen was subsequently sent to Dr. Jesse Edwards, a cardiac pathologist, for detailed examination. He also was not able to identify a specific cause of death.

Accidental/traumatic

There were five patients in this category. Four died after operation of accidents and one was murdered 16 years after operation during a gas station holdup. He had had a normal postoperative heart catheterization.

Two of the fatal accidents involved automobiles 12 and 17 years after operation. Ironically, both had had normal postoperative right heart catheterizations. Also, the patient who died 17 years after operation had been one of the first patients operated on as an infant, with normal hemodynamics measured 9 months later.[18]

A third patient died in a fall 8 years later. The fourth accidental death was bizarre. The victim, 15 years postoperative, had by then become a lawyer. He was basking in his backyard swimming pool when a nearby accident caused high voltage wires to fall into the pool electrocuting him. He also had had a normal postoperative heart catheterization.

Complete Heart Block

Four patients had late deaths from this complication, 7 months, 9 months, 6 years, and 8 years after operation. For all of them, the onset of complete block was evident immediately after they came off bypass. All four initially were treated successfully by a pacemaker and myocardial electrodes. All were weaned from their pacemaker and appeared at discharge to be doing very well clinically despite their slow rates. As already mentioned, these patients were the ones who taught us that every patient remaining in complete block must have a pacemaker.[16]

Reoperations

Two patients died following reoperation:

The first patient had had corrective surgery on April 5, 1955, utilizing cross-circulation. At reoperation on August 11, 1965, to correct residual septal and pulmonic stenosis defects, the left anterior descending coronary artery arising anomalously from the right coronary artery and crossing the right ventricular outflow tract was cut during the ventriculotomy. The artery was buried in adhesions and was not visible. The heart could not be restarted.

This vessel had been seen and avoided at the first operation but had not been charted in her records, a policy that was instituted thereafter.

The other patient was reoperated 3 months after the corrective procedure to close a residual ventricular septal defect with a large shunt and congestive failure. The closure was uneventful, but he remained in congestive failure and succumbed 2.5 months later. At autopsy, the ventricular defect was seen to be completely closed, and there was a normal-sized pulmonary outflow tract. However, microscopic examination of the lungs showed a diffuse, very severe occlusive intimal proliferation involving virtually all of the arterioles in every section.

This lesion was first described by Rich in 1948,[19] and we have encountered it in other tetralogies.

Congestive Heart Failure

Two patients died from this cause 3 months and 5 years after operation, respectively.

The first patient, a boy aged 17 years, died 3 months after correction. He had had a right-subclavian pulmonary artery anastomosis 10 years earlier. The recurrence of cyanosis and absence of a shunt murmur resulted in a diagnosis of a "closed shunt," and it was not dissected out at operation. At autopsy, it was evident that the upper end of the ventricular septal defect patch had come loose, causing a defect 9 mm × 14 mm. There was an excellent correction of the pulmonic stenosis, and the previous shunt was wide open.

Obviously, all of these contributed to his cardiac failure. This patient should not have died. The open shunt should

TABLE 4. *Cardiac Reoperations in 105 Survivors**

Years after Operation	VSD	RVOTS	VSD + RVOTS	Tricuspid Regurgitation	ASD	Right Ventricular Aneurysm
<5	2†	—	—	—	—	—
5–15	—	—	2‡	1	—	—
>15	—	3	—	—	1	1

* Total = 10 in 9 patients; 1 patient had 2 reoperations.
† One patient died 2.5 months postrepair due to diffuse obstructive pulmonary arteriolar disease.[19]
‡ One patient died at reoperation due to transected anomalous major coronary artery.
VSD = ventricular septal defect.
RVOTS = right ventricular outflow tract stenosis.
ASD = atrial secundum defect.

have been recognized, and the breakdown in communications after discharge was inexcusable.

The second patient, who was 10 years old at operation, died 5 years after operation at her home in Europe. No details have been available.

Suicide

In the two patients, both young women, who committed suicide 17 and 19 years after operation, depression from cardiac problems did not appear to be an issue since both were asymptomatic from their cardiovascular standpoint. Also, one had had a normal postoperative catheterization. In one, the family claimed that foul play was involved, but the final police report was suicide.

Nonfatal Late Complications

Nonfatal late complications are discussed as either: cardiac-related or noncardiac in origin.

Cardiac Reoperations

There have been ten reoperations on nine of the 105 patients followed (Table 4). As indicated already under late mortality, there have been two fatalities, which have been discussed there. With the exception of one other patient, described below, who had had two reoperations, these reoperations have resulted in normal functional anatomy confirmed by recatherization studies in all but one.

Residual Ventricular Defects

Both of the reoperations for ventricular defects occurred relatively early (less than 5 years) because of the deleterious effects of large L–R shunts without protection from pulmonic stenosis.

One patient, a young boy who was reoperated after only 3 months because of congestive failure, had an unusual, but not unknown, condition. He had widespread pulmonary arteriolar obstructions,[19] and this complication has been discussed above under late mortality.

The other patient was reoperated after 4 years at age 13. She was in no distress, but recatherization had shown a 70% L–R shunt and a pulmonary pressure elevated to 70 mmHg. At the original operation, her VSD was found to be moderately large (2.5 cm) and located high underneath the septal leaflet of the tricuspid valve. A comment was made in the operative note at that time that the exposure of the upper portion of the ventricular defect was difficult, although it was thought that a complete closure had been obtained. At reoperation in 1964 the exposure was excellent *via* a right atriotomy with temporary detachment of the septal leaflet insertion.[20]

Residual Ventricular Defects and Outflow Stenosis

Of the two patients with these defects (Table 4), one died and has been discussed already.

The other patient had a 60% L–R shunt with mild to moderate pulmonic stenosis (gradient: 30 mmHg). Although this patient had no discernible symptoms, he was only 15 years old and better hemodynamics were desired. The original outflow roof placed 10.5 years earlier (in 1957) was intact but was replaced by a wider terry cloth Teflon® prosthesis,[21] and the residual leak along the VSD patch rim was repaired with four stitches. His convalescence was uneventful.

Tricuspid Regurgitation

The reoperation for tricuspid regurgitation done 15 years later represented repair of an iatrogenic injury.

Atrial Septal Defect

The atrial secundum defect closure was performed 21 years after the primary repairs for a lesion that had been overlooked.

Right Ventricular Outflow Tract Stenosis (RVOTS)

Three patients were found to have this residual defect (Table 4). One of them was also the patient who had the right ventricular outflow tract aneurysm and has had two reoperations. All three of these cases are informative and are discussed in more detail below:

An infant was operated on on October 26, 1955, at age 8 months as a semiemergency because of daily syncopal and convulsive sei-

zures. His ventricular septal defect was closed by stitches over polyvinyl pledgets. The pulmonary stenosis was treated by infundibulectomy without an outflow roof. The pulmonary valve was bicuspid but not stenotic. At his first postoperative catheterization in early 1958, he was 3 years old. A closed ventricular defect was found, but a residual pulmonary gradient of 53 mmHg was measured. In subsequent heart catheterizations in 1965 and 1972, this gradient progressively increased to above 100 mmHg at rest, and the patient showed decreased exercise tolerance along with an enlarging right ventricle. At reoperation on June 18, 1973, the pulmonary valvular orifice was only 5 mm, and a valvotomy was necessary along with resection of a subvalvar fibromuscular ring to relieve stenosis. No outflow roof was deemed necessary because of the good-sized pulmonary artery. Recatheterization 4 years later found no shunts and a small outflow gradient (17 mmHg).

Comment. At the initial postoperative heart catheterization in 1958, the patient was just 3 years old and had a moderately severe outflow stenosis. We have found that with such a significant degree of stenosis at such a young age outflow does not keep pace with body growth. Without a significant gradient (less than 20 mmHg), adequate growth of the outflow tract does occur. (Physiologically, the most potent stimulus to the growth of arteries is flow.)

The next patient is this category of severe residual outflow stenosis had her corrective surgery on February 1, 1956, at the age of 26 months (also before the outflow roof concept).

At the original operation the ventricular defect was closed, and the outflow stenosis was severe at both the infundibular and valvar levels. The pulmonary valve was noted to be very thick and nodular. The stenoses were dealt with by the technics then available. At reoperation 17 years later, the stenosis was at the site of this very thick nodular valve and was easily relieved by an outflow roof of terry cloth Teflon.[21]

Comment. Clearly, this patient would have benefited by an outflow root at the first operation.

The third patient has had a complicated history and is the only one in this entire group of 105 patients with a guarded prognosis.

This patient was 11 years old at the time of the correction of his TOF lesions in 1960 by patch closure of his septal defect and a transanular outflow roof of woven Teflon. At reoperation 7 years later (1967), the ventricular defect closure was intact, but there was a large aneurysmal sac originating in the outflow tract. This was excised along with more infundibulum. A new outflow roof of woven Teflon was inserted. There was no sign of a pulmonary valve. The patient recovered and was asymptomatic until a sudden cardiovascular collapse occurred in 1984, apparently due to a ventricular arrhythmia. Recatheterization at that time disclosed residual pulmonary stenosis, severe pulmonary regurgitation, and a coarctation of the right pulmonary artery. At reoperation in 1984 (patient now aged 35 years), the right pulmonary artery coarctation was repaired by arterioplasty, more infundibulum was excised, and a St. Jude prosthetic valve was inserted (without a conduit) at its anatomic site.

Comment. He has had no more arrhythmias and works full time in a strenuous job (foreman in a meat packing plant). However, recatheterization in 1985 with electrophysiologic studies disclosed chronic atrial fibrillation, easily induced ventricular tachycardias, and residual right ventricular hypertension (85 mmHg). There was no gradient over the outflow tract (and valve). However, the proximal right pulmonic arterial pressure was 85/25 and distal right was 50/18 mmHg. This moderate pulmonary hypertension appears to be due to high vascular resistance.

Electrocardiographic Findings and Late Arrhythmias

After correction, each patient has shown a pattern of complete right bundle branch block. Because of its presence, we could not diagnose electrocardiographically coexistent ventricular hypertrophy. Occasionally, T waves showed a pattern of elevated right ventricular systolic pressures. Since we have not routinely performed 24-hour continuous electrocardiogram monitoring in these patients, we do not have that type of information regarding possible arrhythmias. Left hemiblock has been present in several patients and has not been an ominous finding to date.

In addition to the third patient described above under reoperations for RVOTS, there have been four others who have had arrhythmias sufficiently severe to require treatment.

Three of them had tachycardias of various types (supraventricular/ventricular) that have responded well to treatment. One was the patient with tricuspid regurgitation requiring reoperation. It is of interest that the other two showed none of the incremental risk factors that some have implicated as causes of arrhythmias, such as older age at operation, residual right ventricular hypertension, or previous Potts shunts.[22]

Both were operated on at 7 years of age, had not had prior palliative surgery, and had had an uneventful convalescence. Also, both have had routine right heart catheterizations approximately 1 year after operation and had no residual shunts. Right ventricular outflow gradients were measured at 8 mmHg and 14 mmHg respectively. Their arrhythmic symptoms came on later in life for no identifiable reasons and have not limited their life styles in any way. One of these two patients is a woman and has had one pregnancy with a normal delivery and child.

The fifth patient in this category is the only one in this series known to have had the late onset (18 years) of complete heart block with an Adams–Stokes event. This cardiac arrest occurred in September 1975 in her doctor's office where she had gone because of the recent onset of some unusual symptoms. Resuscitation was successful without sequelae, and she has had an implanted pacemaker now for 11 years.

Her prior history is of interest. Corrective surgery was performed on August 25, 1957, when she was 10 years old. Her ventricular defect was closed with a patch. Pulmonary valvotomy and infun-

TABLE 5. *Late Noncardiac, Nonfatal Complications Observed*

Patient	Sex	Date of Operation	Age at Operation (Yr)	Complication	Age at Onset (Yr)	Treatment	Current Status
1	F	12/3/57	15	Breast cancer	41	Mastectomy with implant	Free of cancer at 3 yr
2	M	7/26/60*	21	Thyroid cancer	37	Thyroidectomy	Free of cancer at 10 yr
3	F	8/21/57	3	Multiple sclerosis	30	Symptomatic	Housewife and mother of 3 children
4	F	10/14/58	5	Diabetes mellitus	19	Controlled	Working full time as store manager
5	F	4/21/60	34	Diabetes mellitus	55	Controlled	Working full time as commercial artist
6	M	3/28/60†	9	Urinary obstruction	24	Transurethral resection	No recurrence

* Previous subclavian–pulmonary artery shunt in 1945.

† Previous subclavian–pulmonary artery shunt in 1953.

dibulectomy corrected the outflow stenosis. No outflow roof was necessary. Complete heart block was noted in the operating room immediately postpump, and she was treated by a pacemaker with myocardial electrodes for 3 days, after which sinus rhythm returned.

Recatherization 1 year after operation disclosed no shunts and a 2 mmHg gradient across the outflow tract. She subsequently married and had two normal children by normal deliveries in 1966 and 1973. Another recatheterization in 1975 (2 months after the pacemaker) again showed excellent hemodynamics (no shunt and outflow gradient of 16 mmHg).

Comment. Currently, she leads a very active life, engaging in tennis, golf, and horseback riding regularly.

Bacterial Endocarditis

Only one patient in this series is known to have had this complication, which occurred 3 years after his surgery at the age of 9 years. Treatment was successful. Interestingly, his routine heart catheterization 1 year after operation disclosed excellent findings (no shunt and outflow gradient of 10 mmHg).

Noncardiac, Nonfatal Late Complications Observed

These have occurred in six patients, and details are categorized in Table 5. The occurrence of two cancers seems high, and the radiation received could be a factor, particularly since both organs are in the most likely exposed areas. One of these patients also had a previous shunt at the age of 6 years.† Neither of these patients had any postoperative complications that would have augmented the usual radiation exposures.

Postoperative Hemodynamic Studies

Beginning with the first patients surgically treated, an effort has been made to urge all patients to return a year or more after operation for a routine right heart catheterization. As a result, 62 of these 105 patients (59%) have undergone one or more hemodynamic studies done during this follow-up (Table 6).

TABLE 6. *Classification of Results of Intracardiac Repair According to Postoperative Hemodynamic Findings*

Classification	Hemodynamic Results	Patients N(%)
Excellent	RVP: <40 mmHG and RV–PA gradient: <20 mmHG and No ventricular shunt*	34 (55)
Good	RVP: 40–60 mmHg or RV–PA gradient: 20–40 mmHg or Small L–R ventricular shunt Qp: Qs <1.5:1	20 (32)
Satisfactory	RVP: 60–70 mmHg or RV–PA gradient: <50 mmHg or Small L–R ventricular shunt Qp: Qs <1.5:1	4 (6.5)
Unsatisfactory	RVP: >70 mmHg RV–PA Gradient: >50 mmHg or L–R ventricular shunt Qp: Qs >1.5:1 or Pulmonary vascular disease	4 (6.5)
Total		62 (100.0)

* Forty-seven of 62 patients (76%) had no shunts; another five patients (8%) had a ventricular shunt detectable only by radioisotope technics (shunt is less than 20%).

RVP = right ventricular pressure.

PA = main pulmonary artery pressure.

† Performed in 1945 by Dr. A. Blalock at Johns Hopkins Hospital, Baltimore, Maryland.

TABLE 7. *Pregnancies, Live Births, and Incidence of Congenital Heart Disease*

Tetralogy Correction*	N	Pregnancies	Live Births N(%)	Children with Cardiac Defects N(%)	Type of Lesions
Mother	22	48	43 (89.5)	3 (7)	2 VSD, 1 tetralogy
Father	18	40	39 (97.5)	3 (7.7)	2 pulmonic stenoses†, 1 VSD
Totals	40	88	82 (93.2)	6 (7.3)	

* There were no couples in which both spouses had operations.

† Both from same family.
VSD = ventricular septal defect.

We have classified these results as: *Excellent, Good, Satisfactory,* or *Unsatisfactory,* as detailed in Table 6. When patients have had more than one postoperative heart catheterization, their last study has been utilized for their classification (this applies also to patients reoperated and then recatheterized). If the patient was not recatheterized after reoperation, the preoperative heart catheterization data were used for classification.

Fifty-five per cent, or 34 patients, have been classified *Excellent; i.e.,* they have normal or close to normal hemodynamics (Table 6). In addition, another 32%, or 20 patients, are classified *Good* with a near-normal hemodynamics measured. Thus, 54 patients, or 86% of the total studied, have had hemodynamics consistent with a normal or very close to normal life expectancy. Even the *Satisfactory* group of four patients (6.5%) do not warrant reoperation but are being observed more closely over the years to detect any signs of cardiac dysfunction. In general, the *Unsatisfactory* group needs reoperation when feasible. One other patient in the group has been reoperated but has not been recatheterized. The other two are doing so well clinically with a residual ventricular septal defect and a counterbalancing pulmonic stenosis that they are being observed at present.

Seventy-six per cent (47 of 62) of these patients have no residual shunts, and another 8% (5 patients) have hemodynamically insignificant shunts (Table 6).

The information about the right ventricular outflow tract derived from serial catheterization has indicated unequivocally that the pulmonary outflow tract growth has kept pace with body growth in the absence of residual stenosis. For example, when recatheterization in an infant or young child disclosed an outflow gradient of 50 mmHg or more, that gradient invariably increased to 100 mmHg or more by the early teens. On the other hand, similar patients with an initial gradient of up to 20–25 mmHg have showed no progression over time.

The hemodynamic observations in this patient cohort confirm a previous study of ours on 69 TOF patients catheterized 1–7 years after operation.[23] In that presentation, we also discussed the relevancy of certain aspects of surgical technic to good hemodynamic results. Only one patient in this present group has had evidence of serious pulmonary regurgitation. However, inserting a well-functioning St. Jude prosthetic valve in this patient did not alter his hemodynamic findings appreciably.

Lastly, we found a poor correlation between pressures measured at the time of operation and the ultimate longer-term measurements. This should not be surprising because of the many factors influencing cardiac output, either up or down, in the immediate postpump interval.

Quality of Life among the Long-term Survivors

We believe that the quality of life of these patients can be accurately assessed by looking carefully at three aspects of their lifestyles, namely, pregnancies and parenthood, educational attainments, and occupational achievements.

Pregnancies and Children

There were 40 patients who married and had children during this 26–31 year follow-up. There were 88 pregnancies recorded with 82 (93.2%) live births. Among the live births, there were six infants with congenital cardiac lesions (7.3%), as noted in Table 7.

This incidence of cardiac defects was considerably higher than the expected occurrence in the normal populations of about 1%. However, conversely, 92.7% of the infants were born with normal hearts, and all of the six cardiac lesions observed in this series were of the easily repairable types. There appeared to be surprisingly little difference between which parent had had the corrective surgery (Table 7).

The correction of TOF defects produced a vast improvement in the incidence of live births, 89.5% in this series, when compared with mothers with untreated TOF who had only 42% live births.[24] The proportion of live births was increased to 72% after palliative shunts.[24] Also, the incidence of congenital heart disease in babies from TOF mothers either untreated or treated by shunts was 14% of the live births.[25] This 14% figure compared with the 7% observed in this study. Also, the development after

birth of the infants from these TOF mothers (untreated or treated by shunts) was seriously impaired, even when no cardiac defects were present.[24]

Educational Attainments and Occupational Achievements

Among the 105 patients initially followed, all who were eligible by age and survival finished high school or its European equivalent (Table 8). Nine patients received diplomas from technical schools, and 15 attended college but did not graduate. Thirty-four have graduated from college, and ten of them went on to complete graduate degrees, including two physicians (one a surgeon who also was the youngest patient operated on at the age of 4 months), two Ph.D.'s, and one lawyer. The five patients receiving masters degrees represented such fields as chemistry, education, psychology, and speech pathology.

Some attained more than one graduate degree, such as the young man who received a B.S. in Aero Engineering (Iowa), then Master of Science in Aero Engineering (Stanford), and finally a Ph.D. (Purdue).

In completing Table 8, we have avoided duplication by counting only the highest degree received. Generally, and as expected, there has been a correlation between educational attainments and employment positions. The patient mentioned above with the advanced degrees in aeronautical engineering is now 47 years old and heads a large research division in this country's largest manufacturer of civilian and military aircraft. However, there are also some notable exceptions, such as one former patient who now owns a large midwest restaurant chain. He went to work immediately after finishing high school.

Current employment information has been available about 76 patients (Table 9). In addition to the professional and managerial/executive positions listed, 12 patients are in teaching, nursing, or music (as entertainers or teachers). Ten former patients list positions in sales, supervisory, or secretarial work, and another ten are homemakers. The 11 skilled and advanced technical positions include laboratory technologist, dietitian, hydrologist, commercial artist, plumber, and computer programmer and repair. Semiskilled laborers include carpenters, electricians, farmers, and manual laborers. Only one individual was unemployed, but not for health reasons.

TABLE 8. *Educational Status—Highest Level Beyond High School*

	Former Patients (N)	
Technical school diplomas	9	
College (1–4 yr)	15	
College graduate	24	32% of 105 survivors
Graduate degree (Masters = 5, M.D. = 2, Ph.D. = 2, LL.B. = 1)	10	
Total	58	

Discussion

This is a late study of a number of important parameters in a group of 105 patients who 26–31 years ago were among the first to have intracardiac correction of the complicated malformation of TOF. We believe that these data present an accurate portrayal, because of the 99% follow-up and the time and care taken to collect this information.

Survival

The actuarial figures for late survival of these patients—80% at 20 years and 77% at 30 years—were most encouraging. The relatively small attrition in their third decade, is also noteworthy (Fig. 1).

Almost 40% of the late deaths were due to noncardiac factors such as trauma, suicide, and cancer, which are equally applicable to the general population. These statistics appear to confirm that these patients have moved into the mainstream of the "normal risks of life." It has often been noted that the most common cause of death in the United States from ages 1 to 40 years is trauma from accidents. Of the 13 cardiac-related late deaths that have occurred (Table 3), almost half can now be prevented by the advances in knowledge and technology. In this context, we refer to those deaths due to heart block and certainly some of those due to congestive failure and reoperation.

The relatively large group of five sudden and unexpected deaths remains an enigma. Our studies have not disclosed any real clues as to who might be vulnerable. Stress electrocardiography and/or routine Holter monitoring might be starting points for a more aggressive investigation of the electrophysiologic mechanisms responsible for triggering late fatal arrhythmias.

Reoperations

In this studied group, the actuarial freedom from reoperations of 97.5% at 10 years, 92.5% at 20 years, and

TABLE 9. *Employment Status*

Occupational Classification	Former Patients (N)
Professional	6
Managerial, executive	15
Teaching, nursing, music	12
Sales, supervisory, secretarial	10
Homemaker	10
Skilled/advanced technical	11
Semiskilled labor	12
Total	76

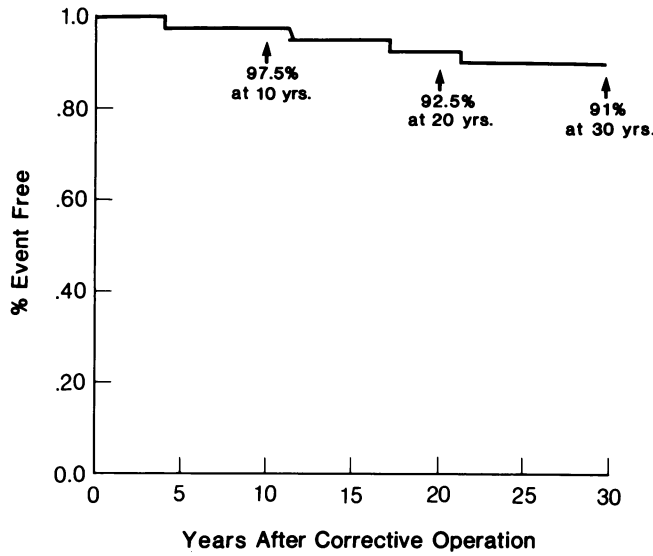

FIG. 2. Actuarial freedom from reoperations after repair of tetrology of Fallot defects.

91% at 30 years has been a very satisfying finding (Fig. 2). These observations, along with the survival observed and the favorable postoperative hemodynamics measured, are all important tests of the degree to which the technical demands of these operations have been recognized and fulfilled.

Another important factor, one often overlooked by others, contributing to this low incidence of later reoperations is the fact that we have never used pericardial patches for septal defect closure or outflow roofs. A pericardial patch always undergoes an aseptic necrosis with subsequent healing by scarring and cicatricial retraction of an unpredictable amount. Such shrinkage contributes to the possibilities for recurrent pulmonic stenosis or for the pulling out of stitches in septal defect patches. Also, during the aseptic necrosis phase, this tissue is weakened, and, if pressure differences are unfavorable, they predispose the patches to tears (in septal defects) or to the formation of aneurysms (in the outflow tract).

Postoperative Hemodynamics

A significantly larger percentage of our patients have had postoperative hemodynamics studies when compared with most other data from late follow-up studies that have been reported. From the beginning of this work, we have made an effort to have all patients undergo a routine right heart catheterization sometime after a year had elapsed.

Apparently, many clinicans have been content to evaluate these patients by clinical assessment. We do not agree. Early in our experience in cardiac surgery for either acquired or congenital lesions, we were impressed by the wide discrepancy sometimes existing between an apparent excellent clinical status and surprisingly poor measured hemodynamics. We have found no good substitute (until the recent advent of Doppler-echocardiographic studies) for catheterization information in advising individual patients on their life expectancy, occupational choices, marriage, pregnancies, or the amount of strenuous physical activity they can tolerate. Also, in a few instances, we were able to advise and carry out a much needed reoperation before significant myocardial disability had occurred. Another important bonus is the value of measured hemodynamics as an early objective test of the surgical methods used.

It is our judgment that patients classified *Excellent* or *Good* (Table 6) will likely have normal or very near to normal life expectancies from their cardiac standpoint.

Childbearing

The data from this study indicated that there is no reason for these patients not to have children if they so desire. Labor and delivery have been well tolerated, and the small increase in the risk of a child with congenital heart disease is offset by the fact that almost all of the possible lesions are readily correctable at an early age, which reduced both the emotional and financial burdens.

Educational and Occupational Attainments

This portion of our study was a distinct and gratifying surprise to us. It is very clear that the educational levels attained by these patients far exceed those of 100 individuals drawn from any level of society, much less a cross section. One third were college graduates, and an impressive number have gone on to advanced positions in medicine, science, engineering, and the law.

It is very intriguing to speculate on possible causes for these unusual findings. Possibly, motivation from having experienced a serious threat to life relatively early followed by recovery might have been a factor in some, but that can hardly be an adequate explanation for all. Doubtless, there are multiple factors involved, but we can safely conclude that their cardiac reparative operations, as major as they were, did not inhibit their goals and aspirations in any way.

Conclusions

One hundred five of 106 patients having intracardiac repairs of TOF defects from 1954 through 1960 who were discharged from the hospital have been followed for 26--31 years. This follow-up was 99% complete in 1986. Twenty-one deaths occurred, giving actuarial survivals of 92.5% at 10 years, 80% at 20 years, and 77% at 30 years. These impressive survival statistics notwithstanding, almost half of the late cardiac-related deaths are now avoidable by advances in knowledge and technology. Among

the 84 long-term survivors, there are few health problems. They have achieved an excellent record for hemodynamics, education, employment, parenthood, and family life.

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Discussion

Dr. Harris B. Shumacker, Jr. (Bethesda, Maryland): Certainly Dr. Lillehei and his colleagues are to be heartily congratulated. I remember, and I am sure everyone here who was there at that time remembers well, the excitement that was caused by his innovative presentation and also the provocative discussion that followed it.

I would like to mention something that is of considerable historic importance but of no practical importance in 1986. It is an event mentioned briefly in the literature in a discussion by Dr. Bill Scott and also even more briefly by Dr. Alfred Blalock.

A year prior to the first operation with cardiopulmonary bypass to which Dr. Lillehei made reference, Dr. Scott in a very hurried operation under hypothermia with a period of only 7 minutes of venacaval occlusion repaired a tetralogy of Fallot with a good result, the first case as far as is known of the intracardiac repair of tetralogy of Fallot. Dr. Scott tells me that there was overriding of the aorta, that the ventricular defect was small and easy to close, and that the infundibular stenosis was corrected. The patient did well for 24 years and then had recurrence of some infundibular stenosis, which I understand required correction.

This operation, which took place 33 years ago, I thought might be of some interest in reference to the modern era that was introduced by Dr. Lillehei and his colleagues from Minneapolis.

Dr. Frank C. Spencer (New York, New York): I rise to express the appreciation of all cardiovascular surgeons for the major contributions of Dr. Lillehei and his group for their pioneering work of developing the corrective operation for tetralogy of Fallot. Both the concept and the performance of the corrective operation were major contributions. At that time (1954), I was in residency training at Johns Hopkins and well recall the astonishment and excitement when the first successful operation was reported.

This contribution was typical of several major discoveries that came from Dr. Lillehei's laboratories between 1950 and 1955, representing many of the cornerstones that launched the new field of open heart surgery. These included the tetralogy operation; the treatment of heart block with a pacemaker; and the discovery of the bubble oxygenator, which enabled many institutions to proceed with clinical surgery.